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SEARCH REQUEST FORM

Requester's Full Name: Thomas S. Heard Examiner #: 80941 Date: 6/6/08

Art Unit: 1654 Phone Number: 2-2064 Serial Number: 10/540,431

Location (Bldg/Room#): REM (Mailbox #): 3C18 Results Format Preferred (circle): PAPER DISK

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To ensure an efficient and quality search, please attach a copy of the cover sheet, claims, and abstract or fill out the following:

Title of Invention: Peptides that Bind to the Heparin Binding Domain of VEGF...

Inventors (please provide full names): See attached BIB sheet

Earliest Priority Date: See attached Bib sheet

Search Topic:

Please provide a detailed statement of the search topic, and describe as specifically as possible the subject matter to be searched. Include the elected species or structures, keywords, synonyms, acronyms, and registry numbers, and combine with the concept or utility of the invention. Define any terms that may have a special meaning. Give examples or relevant citations, authors, etc., if known.

For Sequence Searches Only *Please include all pertinent information (parent, child, divisional, or issued patent numbers) along with the appropriate serial number.*

Please search the sequences in claim 2, SEQ ID NO 1-10.

Please seaurch the generic of claim 1.

Thanks

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263302

STN Registry Search

Inventor search history

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=> d his L10

     (FILE 'HCAPLUS' ENTERED AT 08:32:32 ON 12 JUN 2008)
L10          21 S L6-L9

=> d que L10
L6           21 SEA FILE=HCAPLUS ABB=ON  PLU=ON  ("KULSETH M A"/AU OR "KULSETH
                MARI A"/AU OR "KULSETH MARI ANN"/AU)
L7            9 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L6 AND GE?/CO,CS,PA,SO
L8            2 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L6 AND HEALTH?/CO,CS,PA,SO
L9            2 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L6 AND (HEPARIN OR "VEGF" OR
                "VEGFR-2" OR (VEGFR(W)2) OR (PEPTID?(3A)(BIND? OR DOMAIN OR
                REGION)))
L10          21 SEA FILE=HCAPLUS ABB=ON  PLU=ON  (L6 OR L7 OR L8 OR L9)


=> d his L24

     (FILE 'MEDLINE, BIOSIS, EMBASE, DRUGU' ENTERED AT 08:43:24 ON 12 JUN 2008)
L24          30 S L17-L23
                SAVE TEMP L24 HEA431MLIN/A

     FILE 'USPATFULL' ENTERED AT 08:50:54 ON 12 JUN 2008
                SAVE TEMP L13 HEA431MLSQ/A

=> d que L24
L6           21 SEA FILE=HCAPLUS ABB=ON  PLU=ON  ("KULSETH M A"/AU OR "KULSETH
                MARI A"/AU OR "KULSETH MARI ANN"/AU)
L7            9 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L6 AND GE?/CO,CS,PA,SO
L8            2 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L6 AND HEALTH?/CO,CS,PA,SO
L9            2 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L6 AND (HEPARIN OR "VEGF" OR
                "VEGFR-2" OR (VEGFR(W)2) OR (PEPTID?(3A)(BIND? OR DOMAIN OR
                REGION)))
L16          50 SEA L6
L17          27 SEA L7
L18           1 SEA L8
L19           1 SEA L9
L20           2 SEA L16 AND (HEPARIN OR PEPTID? OR ANALOG?)
L21           0 SEA L16 AND ("VEGF" OR "VEGFR-2" OR (VEGFR(W) 2))
L22           1 SEA L16 AND AMERSHAM?/CO,CS,PA,SO
L23           0 SEA L16 AND (BIND?(3N)(DOMAIN OR REGION OR SITE OR EPITOP?))
L24          30 SEA (L17 OR L18 OR L19 OR L20 OR L21 OR L22 OR L23)


=> dup rem L10 L24
FILE 'HCAPLUS' ENTERED AT 08:52:20 ON 12 JUN 2008
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FILE 'BIOSIS' ENTERED AT 08:52:20 ON 12 JUN 2008
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PROCESSING COMPLETED FOR L10
PROCESSING COMPLETED FOR L24
L25 22 DUP REM L10 L24 (29 DUPLICATES REMOVED)
ANSWERS '1-21' FROM FILE HCAPLUS
ANSWER '22' FROM FILE BIOSIS

Inventor search results

=> d L25 1-22 ibib ab

L25 ANSWER 1 OF 22 HCAPLUS COPYRIGHT 2008 ACS on STN DUPLICATE 1
ACCESSION NUMBER: 2008:515002 HCAPLUS Full-text
TITLE: Characterization of novel mutations in the catalytic domain of the PCSK9 gene
AUTHOR(S): Cameron, J.; Holla, Oe. L.; Laerdahl, J. K.; Kulseth, M. A.; Ranheim, T.; Rognes, T.; Berge, K. E.; Leren, T. P.
CORPORATE SOURCE: Department of Medical Genetics, Medical Genetics Laboratory, University of Oslo, Oslo, Norway
SOURCE: Journal of Internal Medicine (2008), 263(4), 420-431
CODEN: JINMEO; ISSN: 0954-6820
PUBLISHER: Blackwell Publishing Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Objectives. To expand our understanding of the structure and function of proprotein convertase subtilisin/kexin type 9 (PCSK9) by studying how naturally occurring mutations in PCSK9 disrupt the function of PCSK9. Design. Mutations in PCSK9 were identified by sequencing of DNA from subjects with hypo- or hypercholesterolemia. The effect of the identified mutations on the autocatalytic cleavage and secretion of PCSK9, as well as the effect on PCSK9-mediated degradation of the low d. lipoprotein receptors, were determined in HepG2 or HEK293 cells transiently transfected with mutant PCSK9-containing plasmids. The findings were collated to the clin. characteristics of the subjects possessing these mutations, and the phenotypic effects were analyzed in terms of available structural data for PCSK9. Results. Five novel mutations in PCSK9 were identified. Mutation R215H was a gain-of-function mutation which causes hypercholesterolemia. Mutation G236S and N354I were loss-of-function mutations due to failure to exit the endoplasmic reticulum or failure to undergo autocatalytic cleavage, resp. Mutations A245T and R272Q were most likely normal genetic variants. By comparing the number of patients with gain-of-function mutations in PCSK9 with the number of familial hypercholesterolemia heterozygotes among subjects with hypercholesterolemia, the prevalence of subjects with gain-of-function mutations in PCSK9 in Norway can be estimated to one in 15 000. Conclusion. This study has provided novel information about the structural requirements for the normal function of PCSK9. However, more studies are needed to determine the mechanisms by which gain-of-function mutations in PCSK9 cause hypercholesterolemia.

L25 ANSWER 2 OF 22 HCAPLUS COPYRIGHT 2008 ACS on STN DUPLICATE 2
ACCESSION NUMBER: 2007:503085 HCAPLUS Full-text
DOCUMENT NUMBER: 146:454780
TITLE: 4-phenylbutyrate restores the functionality of a misfolded mutant low-density lipoprotein receptor
AUTHOR(S): Tveten, Kristian; Holla, Oeystein L.; Ranheim, Trine; Berge, Knut E.; Leren, Trond P.; Kulseth, Mari A.
CORPORATE SOURCE: Medical Genetics Laboratory, Department of Medical Genetics, Rikshospitalet-Radiumhospitalet Medical Center, Oslo, Norway
SOURCE: FEBS Journal (2007), 274(8), 1881-1893
CODEN: FJEOAC; ISSN: 1742-464X
PUBLISHER: Blackwell Publishing Ltd.

DOCUMENT TYPE: Journal
LANGUAGE: English
AB Familial hypercholesterolemia is an autosomal dominant disease caused by mutations in the gene encoding the low-d. lipoprotein receptor. To date, more than 900 different mutations have been described. Transport-defective mutations (class 2) causing partial or complete retention of the receptor in the endoplasmic reticulum are the predominant class of mutations. In a cell culture system (Chinese hamster ovary cells), we show that chemical chaperones are able to mediate rescue of a transport-defective mutant (G544V), and that the ability to obtain rescue is mutation dependent. In particular, the low mol. mass fatty acid derivative 4-phenylbutyrate mediated a marked increase in the transport of G544V-mutant low-d. lipoprotein receptor to the plasma membrane. Thirty per cent of the mutant receptor was able to escape from the endoplasmic reticulum and reach the cell surface. The rescued receptor had reduced stability, but was found to be as efficient as the wild-type low-d. lipoprotein receptor in binding and internalizing low-d. lipoprotein. In addition to 4-phenylbutyrate, we also studied 3-phenylpropionate and 5-phenylvalerate, and compared their effect on rescue of the G544V-mutant low-d. lipoprotein receptor with their ability to increase overall gene expression caused by their histone deacetylase inhibitor activity. No correlation was found. Our results indicate that the effect of these agents was not solely mediated by their ability to induce gene expression of proteins involved in intracellular transport, but rather could be due to a direct chemical chaperone activity. These data suggest that rescue of mutant low-d. lipoprotein receptor is possible and that it might be feasible to develop pharmacol. chaperones to treat familial hypercholesterolemia patients with class 2 mutations.
REFERENCE COUNT: 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L25 ANSWER 3 OF 22 HCAPLUS COPYRIGHT 2008 ACS on STN DUPLICATE 3
ACCESSION NUMBER: 2006:375248 HCAPLUS Full-text
DOCUMENT NUMBER: 145:56868
TITLE: Effect of mutations in the PCSK9 gene on the cell surface LDL receptors
AUTHOR(S): Cameron, Jamie; Holla, Oystein L.; Ranheim, Trine; Kulseth, Mari Ann; Berge, Knut Erik; Leren, Trond P.
CORPORATE SOURCE: Medical Genetics Laboratory, Department of Medical Genetics, Rikshospitalet University Hospital, Oslo, N-0027, Norway
SOURCE: Human Molecular Genetics (2006), 15(9), 1551-1558
CODEN: HMGEE5; ISSN: 0964-6906
PUBLISHER: Oxford University Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The proprotein convertase subtilisin/kexin type 9 (PCSK9) gene is involved in the post-transcriptional regulation of the low-d. lipoprotein (LDL) receptors (LDLR). Mutations in the PCSK9 gene have been associated with both hypocholesterolemia and hypercholesterolemia through 'loss-of-function' and gain-of-function' mechanisms, resp. We have studied the effect of the four loss-of-function mutations R46L, G106R, N157K and R237W and the two gain-of-function mutations S127R and D374Y on the autocatalytic activity of PCSK9, as well as on the amount of the cell surface LDLR and internalization of LDL in transiently transfected HepG2 cells. The two groups of mutations did not differ with respect to autocatalytic activity of PCSK9, but they did differ with respect to the amount of cell surface LDLR and internalization of LDL. The four loss-of-function mutations had a 16% increased level of cell surface LDLR and a 35% increased level of internalization of LDL as compared with WT-

PCSK9. The two gain-of-function mutations had a 23% decreased level of cell surface LDLR and a 38% decreased level of internalization of LDL as compared with WT-PCSK9. Our studies have also shown that transfer of media from transiently transfected HepG2 cells to untransfected HepG2 cells, reduces the amount of cell surface LDLR and internalization of LDL in the untransfected cells within 20 min of media transfer. Thus, PCSK9 or a factor acted upon by PCSK9, is secreted from the transfected cells and degrades LDLR both in transfected and untransfected cells.

REFERENCE COUNT: 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L25 ANSWER 4 OF 22 HCAPLUS COPYRIGHT 2008 ACS on STN DUPLICATE 4
ACCESSION NUMBER: 2006:792248 HCAPLUS Full-text
DOCUMENT NUMBER: 145:434223
TITLE: Model system for phenotypic characterization of sequence variations in the LDL receptor gene
AUTHOR(S): Ranheim, Trine; Kulseth, Mari Ann; Berge, Knut Erik; Leren, Trond Paul
CORPORATE SOURCE: Department of Medical Genetics, Rikshospitalet-Radiumhospitalet Medical Center, Oslo, N-0027, Norway
SOURCE: Clinical Chemistry (Washington, DC, United States) (2006), 52(8), 1469-1479
CODEN: CLCHAU; ISSN: 0009-9147
PUBLISHER: American Association for Clinical Chemistry
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Background: Sequence variations in the LDL receptor (LDLR) gene cause defects of LDLR protein production and function through different mol. mechanisms. Here we describe a cell model system for the phenotypic characterization of sequence variations in the LDLR gene. Well-known sequence variations belonging to LDLR classes 2 to 5 (p.G565V, p.I161D, p.Y828C, and p.V429M) were studied in CHO and HepG2 cells. Methods: Expression of LDLR protein on the cell surface was detected by use of fluorescence-conjugated antibodies against the LDLR and the LDLR activity was measured by incubating the cells with fluorescently labeled and radiolabeled LDL. The intracellular locations of the LDLR mutants and wild-type were also investigated. Results: The class 2A p.G565V sequence variant exhibited an intracellular distribution of LDLR with no active receptors on the cell surface. Both the class 3 p.I161D and class 4 p.Y828C sequence variants gave surface staining but had a reduced ability to bind or internalize LDL, resp. By determining the intracellular locations of the receptors we were able to visualize the accumulation of the class 5 p.V429M sequence variant in endosomes by means of a specific marker, as well as confirming that the class 4 p.Y828C variant was not localized in clathrin-coated pits. Flow cytometry allowed us quant. to determine the amount and activity of receptors. To confirm the results of binding and cell association of fluorescently labeled LDL analyzed by flow cytometry, assays using 125I-labeled LDL were performed. In addition to a useful and valid alternative to radiolabeled LDL, the unique properties of fluorescently labeled LDL allowed a variety of detection technologies to be used. Conclusions: This new approach enables phenotypic characterization of sequence variations in the LDLR gene. The assays developed may be valuable for confirming the pathogenicity of novel missense sequence variations found throughout the LDLR gene.

REFERENCE COUNT: 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L25 ANSWER 5 OF 22 HCAPLUS COPYRIGHT 2008 ACS on STN DUPLICATE 5
ACCESSION NUMBER: 2006:12395 HCAPLUS Full-text
DOCUMENT NUMBER: 144:210610
TITLE: Retention of Mutant Low Density Lipoprotein Receptor

in Endoplasmic Reticulum (ER) Leads to ER Stress
AUTHOR(S): Sorensen, Stine; Ranheim, Trine; Bakken, Kari Solberg; Leren, Trond P.; Kulseth, Mari Ann
CORPORATE SOURCE: Medical Genetics Laboratory, Department of Medical Genetics, Rikshospitalet, University Hospital, Oslo, N-0027, Norway
SOURCE: Journal of Biological Chemistry (2006), 281(1), 468-476
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Familial hypercholesterolemia is an autosomal dominant disease caused by mutations in the gene encoding the low d. lipoprotein receptor (LDLR). More than 50% of these mutations lead to receptor proteins that are completely or partly retained in the endoplasmic reticulum (ER). The mechanisms involved in the intracellular processing and retention of mutant LDLR are poorly understood. In the present study we show that the G544V mutant LDLR assocs. with the chaperones Grp78, Grp94, ERp72, and calnexin in the ER of transfected Chinese hamster ovary cells. Retention of the mutant LDLR was shown to cause ER stress and activation of the unfolded protein response. We observed a marked increase in the activity of two ER stress sensors, IRE1 and PERK. These results show that retention of mutant LDLR in ER induces cellular responses, which might be important for the clin. outcome of familial hypercholesterolemia.

REFERENCE COUNT: 51 THERE ARE 51 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L25 ANSWER 6 OF 22 HCAPLUS COPYRIGHT 2008 ACS on STN DUPLICATE 6
ACCESSION NUMBER: 2006:751695 HCAPLUS Full-text
DOCUMENT NUMBER: 146:160746
TITLE: Low-density lipoprotein receptor activity in Epstein-Barr virus-transformed lymphocytes from heterozygotes for the D374Y mutation in the PCSK9 gene
AUTHOR(S): Holla, Oe. L.; Cameron, J.; Berge, K. E.; Kulseth, M. A.; Ranheim, T.; Leren, T. P.
CORPORATE SOURCE: Medical Genetics Laboratory, Department of Medical Genetics, Rikshospitalet University Hospital, Oslo, Norway
SOURCE: Scandinavian Journal of Clinical and Laboratory Investigation (2006), 66(4), 317-328
CODEN: SJCLAY; ISSN: 0036-5513
PUBLISHER: Taylor & Francis
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Objective. Missense mutations in the proprotein convertase subtilisin/kexin type 9 (PCSK9) gene have been found to cause autosomal dominant hypercholesterolemia. The objective of this study was to investigate possible mechanisms by which mutation D374Y in the PCSK9 gene causes hypercholesterolemia. Material and methods. Binding and internalization of low-d. lipoprotein LDL in Epstein-Barr virus (EBV)-transformed lymphocytes from D374Y heterozygotes were examined The autocatalytic activity of the D374Y mutant was studied in transiently transfected HEK293 cells. Results. As determined by Western blot anal. of transiently transfected HEK293 cells, the autocatalytic activity of the D374Y mutant was .apprx.95 % of the wild-type. Levels of PCSK9 mRNA in EBV-transformed lymphocytes from D374Y heterozygotes and normal controls were similar and less than 1/1000 of the level in HepG2 cells. The amount of cell surface LDL receptors (LDLRs) in EBV-transformed lymphocytes from five D374Y heterozygotes was non-significantly increased by

17 % compared with the amount in normal controls. LDLR-dependent binding and internalization of LDL in EBV-transformed lymphocytes from D374Y heterozygotes were non-significantly reduced by 11 % and 12 %, resp., compared to the corresponding values in normal controls. Conclusions. LDLR-mediated endocytosis of LDL is not reduced in EBV-transformed lymphocytes from D374Y heterozygotes. Because of the extremely low levels of PCSK9 mRNA in EBV-transformed lymphocytes, it is possible that the LDLR-dependent endocytosis of LDL could be more severely affected in hepatocytes from D374Y heterozygotes than in EBV-transformed lymphocytes.

REFERENCE COUNT: 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L25 ANSWER 7 OF 22 HCAPLUS COPYRIGHT 2008 ACS on STN DUPLICATE 7

ACCESSION NUMBER: 2006:910895 HCAPLUS Full-text

DOCUMENT NUMBER: 145:500643

TITLE: Analysis of alternatively spliced isoforms of human LDL receptor mRNA

AUTHOR(S): Tveten, Kristian; Ranheim, Trine; Berge, Knut Erik; Leren, Trond P.; Kulseth, Mari Ann

CORPORATE SOURCE: Medical Genetics Laboratory, Department of Medical Genetics, Rikshospitalet-Radiumhospitalet Medical Center, Oslo, N-0027, Norway

SOURCE: Clinica Chimica Acta (2006), 373(1-2), 151-157
CODEN: CCATAR; ISSN: 0009-8981

PUBLISHER: Elsevier Ltd.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Background: The low d. lipoprotein receptor (LDLR) family is a family of structurally related cell surface receptors with conserved exon/intron organization. Several members of this family have been shown to undergo alternative splicing. However, no alternative splicing of the LDLR pre-mRNA has so far been described. Methods: In the present study alternative splicing of human LDLR pre-mRNA has been studied in eight different tissues and four different cell lines using reverse transcription (RT) PCR. A quant. real-time PCR with exon-exon boundary spanning primers was established to measure the relative amount of two novel isoforms. Results: Several novel isoforms were identified by RT-PCR of which the isoforms lacking exon 4 or 12 were two of the most prominent. Although highly detectable by RT-PCR, the quantification by real-time PCR revealed low levels of these isoforms. Conclusions: Novel isoforms of LDLR mRNA are described. Quantification by real-time PCR of two of the alternatively spliced isoforms revealed low amount of these isoforms in the examined tissues and cell lines. Further investigations are needed to evaluate if these isoforms represent functional transcripts of LDLR mRNA.

REFERENCE COUNT: 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L25 ANSWER 8 OF 22 HCAPLUS COPYRIGHT 2008 ACS on STN DUPLICATE 8

ACCESSION NUMBER: 2002:146449 HCAPLUS Full-text

DOCUMENT NUMBER: 136:292070

TITLE: Reduced secretion of triacylglycerol in CaCo-2 cells transfected with intestinal fatty acid-binding protein

AUTHOR(S): Gedde-Dahl, Ane; Kulseth, Mari Ann; Ranheim, Trine; Drevon, Christian A.; Rustan, Arild C.

CORPORATE SOURCE: Department of Pharmacology, School of Pharmacy, University of Oslo, Oslo, N-0316, Norway

SOURCE: Lipids (2002), 37(1), 61-68
CODEN: LPDSAP; ISSN: 0024-4201

PUBLISHER: AOCS Press

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The fatty acid-binding proteins are hypothesized to be involved in cellular fatty acid transport and trafficking. We established CaCo-2 cells stably transfected with intestinal fatty acid-binding protein (I-FABP) and examined how the expression of this protein may influence fatty acid metabolism I-FABP expression was detectable in I-FABP-transfected cells, whereas parent CaCo-2 cells as well as mock-transfected cells failed to express detectable levels of I-FABP mRNA or protein at any stage of differentiation. For studies of lipid metabolism, cells were incubated with [14C]oleic acid in taurocholate micelles containing monoolein, and distribution of labeled fatty acid in cellular and secreted lipids was examined In one transfected cell clone, expressing the highest level of I-FABP, labeled cellular triacylglycerol increased approx. twofold as compared to control cells. The level of intracellular triacylglycerol in two other I-FABP-transfected clones resembled that of control cells. However, secretion of triacylglycerol was markedly reduced in all the I-FABP-expressing cell lines. Our data suggest that increased expression of I-FABP leads to reduced triacylglycerol secretion in intestinal cells.

REFERENCE COUNT: 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L25 ANSWER 9 OF 22 HCAPLUS COPYRIGHT 2008 ACS on STN DUPLICATE 9

ACCESSION NUMBER: 1999:600983 HCAPLUS Full-text

DOCUMENT NUMBER: 131:285203

TITLE: Stimulation of serglycin and CD44 mRNA expression in endothelial cells exposed to TNF-α and IL-1α

AUTHOR(S): Kulseth, Mari Ann; Kolset, Svein Olav; Ranheim, Trine

CORPORATE SOURCE: Institute for Nutrition Research, Faculty of Medicine, University of Oslo, Oslo, N-0316, Norway

SOURCE: Biochimica et Biophysica Acta, General Subjects (1999), 1428(2-3), 225-232
CODEN: BBGSB3; ISSN: 0304-4165

PUBLISHER: Elsevier B.V.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Serglycin is a widely distributed proteoglycan, previously assumed to be hematopoietic cell specific. However, the results presented show that serglycin mRNA is expressed outside the hematopoietic cell system. High levels of serglycin mRNA were detected in endothelial cells and smooth muscle cells, whereas low levels were detected in skin fibroblasts. To further analyze the importance of serglycin in endothelial cells, the expression of serglycin mRNA was measured following activation of an endothelial cell line derived from human umbilical cord vein (HUV-EC-C), by the proinflammatory cytokines TNF-α and IL-1α. The level of serglycin mRNA increased in a time- and dose-dependent way. TNF-α (7 ng/mL) was the most potent inducer, increasing the level of serglycin mRNA 2.5 times after 24 h of stimulation. Serglycin has been shown to be a ligand for CD44, a membrane protein expressed in endothelial cells. Following stimulation of the endothelial cells, the level of CD44 mRNA also increased. Again, TNF-α (7 ng/mL) turned out to be the most potent inducer, increasing the level of CD44 mRNA 5.5 times after 24 h of stimulation. Both TNF-α and IL-1α stimulation of the endothelial cells resulted in an increase in the total incorporation of [35S]sulfate into macromols., which probably indicates an increase in the total production of proteoglycans. A stimulation of endothelial cells by proinflammatory agents resulted in an increase in both serglycin and CD44 mRNA expression, indicating that serglycin, as well as CD44, may participate in the inflammatory process of leukocyte migration.

REFERENCE COUNT: 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L25 ANSWER 10 OF 22 HCAPLUS COPYRIGHT 2008 ACS on STN DUPLICATE 10
ACCESSION NUMBER: 1998:329197 HCAPLUS Full-text
DOCUMENT NUMBER: 129:80026
TITLE: Proteoglycans in macrophages: characterization and possible role in the cellular uptake of lipoproteins
AUTHOR(S): Halvorsen, Bente; Aas, Une K.; Kulseth, Mari Ann; Drevon, Christian A.; Christiansen, Erling N.; Kolset, Svein O.
CORPORATE SOURCE: Institute for Nutrition Research, University of Oslo, Oslo, 0316, Norway
SOURCE: Biochemical Journal (1998), 331(3), 743-752
CODEN: BIJOAK; ISSN: 0264-6021
PUBLISHER: Portland Press Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The murine macrophage cell line J774 was incubated with [35S]sulfate. The cell-associated 35S-labeled macromols. were shown to be proteoglycans and glycosaminoglycans in similar amts. The possible presence of cell-surface proteoglycans was investigated by incubating [35S]sulfate-labeled cells with trypsin for 15 min. The released material contained approx. 70 % free glycosaminoglycan chains and 30 % proteoglycans. The latter component was demonstrated by HNO2 treatment to contain heparan sulfate. In the total cell fraction not treated with trypsin a small but significant portion was shown to be chondroitin sulfate proteoglycan. The cell-associated glycosaminoglycans contained both chondroitin sulfate and heparan sulfate. To investigate possible biol. functions of cell-surface proteoglycans in macrophages, cells were incubated with NaClO3 to inhibit sulfation of proteoglycans and β-D-xyloside to abrogate proteoglycan expression. The uptake of oxidized 125I-tyraminylcellobiose-labeled low-d. lipoprotein (125I-TC-LDL) was typically two to three times higher than that of native 125I-TC-LDL in untreated J774 cells. The cellular uptake at 37° of native 125I-TC-LDL was decreased 25 % after both NaClO3 and xyloside treatment, whereas the uptake of oxidized 125I-TC-LDL was decreased 35 % after both types of treatment. The mRNA levels for the scavenger receptor A-II and the LDL receptor were not affected by NaClO3 or xyloside treatment. Furthermore, fluid-phase endocytosis, measured as uptake of horseradish peroxidase, and receptor-mediated endocytosis, measured as uptake of 125I-TC-ovalbumin, were not affected by NaClO3 treatment of J774 cells. Removal of cell-surface chondroitin sulfate with chondroitinase ABC decreased only the binding of native 125I-TC-LDL, whereas removal of heparan sulfate with heparitinase decreased the binding of both oxidized and native 125I-TC-LDL. Addition of lipoprotein lipase increased the uptake of oxidized 125I-TC-LDL 1.7 times and the uptake of native 125I-TC-LDL 2.1 times. The binding of the former was more sensitive to NaClO3 treatment than the latter. The results presented support the notion that some of the uptake pathways for lipoproteins in the foam-cell-forming macrophages depend on the presence of cell-surface heparan sulfate and chondroitin sulfate.
REFERENCE COUNT: 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L25 ANSWER 11 OF 22 HCAPLUS COPYRIGHT 2008 ACS on STN DUPLICATE 11
ACCESSION NUMBER: 1999:11119 HCAPLUS Full-text
DOCUMENT NUMBER: 130:151530
TITLE: Cell proliferation, apoptosis and accumulation of lipid droplets in U937-1 cells incubated with eicosapentaenoic acid
AUTHOR(S): Finstad, Hanne S.; Drevon, Christian A.; Kulseth, Mari Ann; Synstad, Anne V.; Knudsen, Eirunn; Kolset, Svein Olav

CORPORATE SOURCE: Institute for Nutrition Research, University of Oslo, Oslo, 0316, Norway
SOURCE: Biochemical Journal (1998), 336(2), 451-459
CODEN: BIJOAK; ISSN: 0264-6021
PUBLISHER: Portland Press Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The monocytic cell line U937-1 was cultured in the presence of eicosapentaenoic acid (20:5, n-3) (EPA) or oleic acid (18:1, n-9) (OA). EPA caused a dose-dependent inhibition of cell proliferation, whereas OA had no effect. At the highest EPA concns., 120 and 240 μM, inhibition of cell proliferation was accompanied by initiation of apoptosis. A concentration of 60 μM EPA caused a 35% reduction in cell proliferation without inducing apoptosis, and was therefore used for further studies. Addition of antioxidants or inhibitors of eicosanoid synthesis had no influence on the reduced cell proliferation after EPA treatment. The inhibition required continuous presence of EPA in the incubation medium as the cells resumed a normal proliferation rate when they were placed in EPA-free medium. The inhibition of proliferation was not accompanied by differentiation into macrophage-like cells, as expression of serglycin and the ability to perform respiratory burst was unaffected by EPA. Expression of CD23 mRNA increased when the cells were incubated with EPA, but to a smaller extent than after retinoic acid (RA) or PMA treatment. Furthermore, expression of the monocytic differentiation markers CD36 and CD68 was lower in cells treated with EPA or OA when compared with untreated cells. The cell cycle distribution of U937-1 cells was similar in cells incubated with EPA or PMA, whereas RA-treated cells accumulated in the G1 phase. Side scatter increased in cells incubated with EPA and OA, which was ascribed to an accumulation of lipid droplets after examination of the cells by electron microscopy. The number of droplets per cell was higher in cells exposed to EPA than OA. The cellular triacylglycerol (TAG) increased 5.5- and 15.5-fold after incubation with OA and EPA resp. No difference in the cellular content of cholesterol compared with untreated cells was observed The TAG fraction in EPA-treated cells contained high amts. of EPA and docosapentaenoic acid and minor amts. of docosahexaenoic acid, whereas OA-treated cells had high levels of OA in the TAG. In cells incubated with a sulfur-substituted EPA, only minor effects on cell proliferation and no accumulation of cellular TAG were observed These findings may indicate the existence of other mechanisms for regulation of cell behavior by very-long-chain polyunsatd. n-3 fatty acids than the well established lipid peroxide and eicosanoid pathways.

REFERENCE COUNT: 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L25 ANSWER 12 OF 22 HCAPLUS COPYRIGHT 2008 ACS on STN DUPLICATE 12
ACCESSION NUMBER: 1995:17958 HCAPLUS Full-text
DOCUMENT NUMBER: 122:73428
ORIGINAL REFERENCE NO.: 122:13779a,13782a
TITLE: Chromosomal localization and detection of DNA polymorphisms in the bovine polymeric immunoglobulin receptor gene
AUTHOR(S): Kulseth, M A.; Toldo, S Solinas; Fries, R.; Womack, J.; Lien, S.; Rogne, S.
CORPORATE SOURCE: Dep. Anim. Sci., Agric. Univ., as, N-1432, Norway
SOURCE: Animal Genetics (1994), 25(2), 113-17
CODEN: ANGEE3; ISSN: 0268-9146
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Polymeric Ig receptor (PIGR) mediates transcellular transport of secretory antibodies in glandular and mucosal epithelial cells. By use of a bovine-rodent somatic cell hybrid panel the bovine PIGR locus has been assigned to

syntenic group U1. Using in situ hybridization, PIGR was localized to bovine chromosome 16, segment q13, thus confirming the recent assignment of syntenic group U1 to this chromosome. Two common restriction fragment length polymorphisms (RFLPs) with the enzymes BamHI and MspI were detected using the PIGR cDNA as probe. Direct PCR sequencing of a segment in the PIGR coding region (nucleotides 162-413) from 13 bulls of Norwegian Cattle revealed single nucleotide exchanges at two positions. An efficient PCR-RFLP method for detection of these mutations was developed.

L25 ANSWER 13 OF 22 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2004:566645 HCAPLUS Full-text
DOCUMENT NUMBER: 141:117189
TITLE: vascular endothelial growth factor receptor 2 (VEGFR-2)-targeted peptides for therapeutic and diagnostic use, and preparation thereof
INVENTOR(S): Kulseth, Mari Ann
PATENT ASSIGNEE(S): Amersham Health As, Norway
SOURCE: PCT Int. Appl., 24 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004058803	A2	20040715	WO 2003-NO443	20031229
WO 2004058803	A3	20040910		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2003288809	A1	20040722	AU 2003-288809	20031229
EP 1578784	A2	20050928	EP 2003-781115	20031229
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
JP 2006524183	T	20061026	JP 2004-563062	20031229
US 20060135434	A1	20060622	US 2005-540064	20050622
PRIORITY APPLN. INFO.:			NO 2002-6285 A	20021230
			WO 2003-NO443 W	20031229

OTHER SOURCE(S): MARPAT 141:117189
AB The invention discloses peptides for targeting to vascular endothelial growth factor receptor 2 (VEGFR-2). The invention further discloses to their use in therapeutically effective treatment as well as for diagnostic imaging techniques. Peptide preparation is described.

L25 ANSWER 14 OF 22 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2004:566644 HCAPLUS Full-text
DOCUMENT NUMBER: 141:117188
TITLE: Peptides that bind to the heparin-binding domain of VEGF and

VEGFR-2, preparation, and therapeutic and diagnostic use
INVENTOR(S): Kulseth, Mari Ann
PATENT ASSIGNEE(S): Amersham Health As, Norway
SOURCE: PCT Int. Appl., 31 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004058802	A1	20040715	WO 2003-NO444	20031229
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2003288810	A1	20040722	AU 2003-288810	20031229
EP 1578785	A1	20050928	EP 2003-781116	20031229
EP 1578785	B1	20080213		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
JP 2006523608	T	20061019	JP 2004-563063	20031229
AT 386050	T	20080315	AT 2003-781116	20031229
US 20060089307	A1	20060427	US 2005-540431	20050622
PRIORITY APPLN. INFO.:			NO 2002-6286 A	20021230
			WO 2003-NO444 W	20031229

OTHER SOURCE(S): MARPAT 141:117188
AB The invention discloses peptides for targeting that bind to the heparin-binding domain of vascular endothelial growth factor and its receptor vascular endothelial growth factor receptor 2, VEGFR-2. The invention also discloses the use of these peptides in therapeutically effective treatment as well as for diagnostic imaging techniques. Preparation of peptides is described.

L25 ANSWER 15 OF 22 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2001:64189 HCAPLUS Full-text
DOCUMENT NUMBER: 134:128187
TITLE: Method of the identification of a receptor using gas microbubble encapsulated cells expressing specific peptides
INVENTOR(S): Kulseth, Mari Ann; Lovhaug, Dagfinn; Godal, Aslak
PATENT ASSIGNEE(S): Nycomed Imaging AS, Norway
SOURCE: PCT Int. Appl., 18 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

WO 2001006007	A2	20010125	WO 2000-NO245	20000720
WO 2001006007	A3	20010719		
W: JP, NO, US				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
EP 1196555	A2	20020417	EP 2000-948425	20000720
EP 1196555	B1	20070919		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY				
AT 373707	T	20071015	AT 2000-948425	20000720
US 20020106643	A1	20020808	US 2002-52299	20020118
US 6806045	B2	20041019		
PRIORITY APPLN. INFO.:			GB 1999-17111 A	19990721
			US 1999-146865P P	19990803
			WO 2000-NO245 W	20000720

AB A method for the identification and characterization of a receptor in target tissue for which a selected vector has affinity, wherein a transfected cell line expressing the receptor is added to a suspension of encapsulated microbubbles to which the selected vector is coupled and allowing the microbubbles and cells coupled thereto to float to the surface of the suspension. Upon isolating the microbubble-bound cells at the surface, these may be cultured to study the receptor, or cells may be lysed, amplifying the receptor-encoding cDNA and sequencing the cDNA. Specifically, a retroviral cDNA library is created using mRNA isolated from 25 endothelial cells and transfected into the RetroPakT PT67 packaging cell line. A suspension of perfluorobutane microbubbles encapsulated with distearoylphosphatidylserine doped with a lipopeptide comprising an endothelial specific peptide, emerged after phage display biopanning, is added to the transfected cells. Floated microbubble-bound cells are isolated e.g. by flow cytometry. The isolated cells from above are lysed and the cDNA encoding the peptide receptor is amplified by a polymerase chain reaction using the primers provided with the pLNCX retroviral vector. The method may also be used diagnostically to detect the presence of a disease marker in a sample.

L25 ANSWER 16 OF 22 HCAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER:	1998:534518 HCAPLUS Full-text
DOCUMENT NUMBER:	129:228820
TITLE:	Serglycin expression during monocytic differentiation of U937-1 cells
AUTHOR(S):	Kulseth, Mari Ann; Mustorp, Stina Lund; Uhlin-Hansen, Lars; Oberg, Fredrik; Kolset, Svein Olav
CORPORATE SOURCE:	Institute for Nutrition Research, University of Oslo, Oslo, Norway
SOURCE:	Glycobiology (1998), 8(8), 747-753 CODEN: GLYCE3; ISSN: 0959-6658
PUBLISHER:	Oxford University Press
DOCUMENT TYPE:	Journal
LANGUAGE:	English

AB Serglycin (I) is the major proteoglycan in most hematopoietic cells, including monocytes and macrophages. The monoblastic cell line, U937-1, was used to study the expression of I during proliferation and differentiation. In unstimulated proliferating U937-1 cells I mRNA is nonconstitutively expressed. The level of I mRNA was found to correlate with the synthesis of chondroitin sulfate proteoglycan (CSPG). The U937-1 cells were induced to differentiate into different types of macrophage-like cells by exposing the cells to phorbol-12-myristate-13- acetate (PMA), retinoic acid (RA), or vitamin D3 (II). These inducers of differentiation affected the expression of I mRNA in 3 different ways. The initial up-regulation seen in the normally proliferating cells was not observed in PMA-treated cells. In contrast, RA increased the

initial up-regulation, giving a reproducible 6-fold increase in I mRNA level from 4 to 24 h of incubation, compared to a 4-fold increase in the control cells. II had no effect on the expression of I mRNA. The incorporation of [35S]sulfate into CSPG decreased .apprx.50% in all 3 differentiated cell types. Further, the [35S]CSPGs expressed were of larger size in PMA-treated cells than controls, but smaller after RA treatment. This was due to the expression of CSPGs, with CS-chains of 25 and 5 kDa in PMA- and RA-treated cells, resp., compared to 11 kDa in the controls. II had no significant effect on the size of CSPG produced. PMA-treated cells secreted 75% of the [35S]PGs expressed, but the major portion was retained in cells treated with II or RA. The differences seen in I mRNA levels, the macromol. properties of I and in the PG secretion patterns, suggest that I may have different functions in different types of macrophages.

REFERENCE COUNT: 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L25 ANSWER 17 OF 22 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1995:457761 HCAPLUS Full-text
DOCUMENT NUMBER: 122:263179
ORIGINAL REFERENCE NO.: 122:48033a,48036a
TITLE: Cloning and characterization of two forms of bovine polymeric immunoglobulin receptor cDNA
AUTHOR(S): Kulseth, Mari Ann; Krajci, Peter; Myklebost, Ola; Rogne, Sissel
CORPORATE SOURCE: Dep. Anim. Sci., Agric. Univ., Aas, N-1432, Norway
SOURCE: DNA and Cell Biology (1995), 14(3), 251-6
CODEN: DCEBE8; ISSN: 1044-5498
PUBLISHER: Liebert
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The polymeric Ig receptor (transmembrane secretory component) mediates transcellular transport of dimeric IgA and pentameric IgM in glandular and mucosal epithelial cells. CDNAs encoding two forms of the bovine polymeric Ig receptor (pIgR) have been cloned and sequenced. The long form contains 3,527 bp and predicts a single open reading frame of 2,271 bp encoding a protein of 757 bp. The extracellular part contains five Ig-like domains. The shorter form lacks the region from residues 458-1,111 corresponding to Ig-like domains 2 and 3. In Northern blot anal. of various bovine tissues, only the long form of pIgR mRNA was detected. By using the reverse transcription-polymerase chain reaction (RT-PCR), both forms were detected. An alignment of the cytoplasmic tail of the pIgR from bovine, human, rabbit, and rat revealed highly conserved areas that may reflect the importance of these regions for intracellular sorting of the receptor.

L25 ANSWER 18 OF 22 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1994:237287 HCAPLUS Full-text
DOCUMENT NUMBER: 120:237287
ORIGINAL REFERENCE NO.: 120:41853a,41856a
TITLE: Cloning and characterization of the bovine immunoglobulin J chain cDNA and its promoter region
AUTHOR(S): Kulseth, Mari Ann; Rogne, Sissel
CORPORATE SOURCE: Dep. Anim. Sci., Agric. Univ., Aas, N-1432, Norway
SOURCE: DNA and Cell Biology (1994), 13(1), 37-42
CODEN: DCEBE8; ISSN: 1044-5498
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The Ig J (joining) chain plays an important role in the assembly of polymeric Igs (dimeric IgA and pentameric IgM) and in the selective transport of these across epithelial cell layers. The primary structure of the bovine J chain

has been determined by sequencing of three cDNAs. The cDNA has an open reading frame of 471 nucleotides encoding a putative protein of 157 amino acids. The 3' untranslated region consists of 698 nucleotides and a poly(A) tail. The 5' untranslated region and the promoter were isolated from a genomic clone. By comparison with the murine J chain gene, the 5' untranslated region was predicted to be 37 bp, giving the bovine J chain cDNA a total length of 1,206 bp. This size was confirmed by Northern blot anal. of total RNA from colon and mammary gland. The amino acid sequence of the bovine J chain shows extensive homol. with the J chain from human, mouse, rabbit, and bullfrog. Anal. of the J chain secondary structure showed a high propensity for forming β-sheets. An alignment of the predicted secondary structure of the J chain from bovine, human, mouse, rabbit, and bullfrog revealed a highly conserved "β-profile.". The promoter sequence of the bovine J chain gene is presented and shown to contain a conserved interleukin-2 (IL-2)-responsive element previously characterized in the murine J chain gene.

L25 ANSWER 19 OF 22 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1993:466188 HCAPLUS Full-text
DOCUMENT NUMBER: 119:66188
ORIGINAL REFERENCE NO.: 119:11824h,11825a
TITLE: A highly sensitive chromogenic microplate assay for quantification of rat and human plasminogen
AUTHOR(S): Kulseth, Mari Ann; Helgeland, Liv
CORPORATE SOURCE: Dep. Biochem., Univ. Oslo, Oslo, 0316, Norway
SOURCE: Analytical Biochemistry (1993), 210(2), 314-17
CODEN: ANBCA2; ISSN: 0003-2697
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A simple and highly sensitive chromogenic microplate assay for quant. of rat and human plasminogen in plasma samples and subcellular fractions has been developed. The assay is based on a conversion of plasminogen to plasmin, using urokinase as an activator, and a subsequent cleavage of a chromogenic plasmin substrate D-alanyl-L-cyclohexylalanyl-L-lysine-p- nitroanilide-dihydroacetate. The p-nitroaniline being released by the cleavage is then measured at 410 nm with a microplate reader. The assay includes an acidification step to make plasminogen more readily activated to plasmin. The method is suitable for analyses of a large number of samples, measuring plasminogen in the nanogram range (0.5-50 ng/50 μL of sample).

L25 ANSWER 20 OF 22 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1994:573326 HCAPLUS Full-text
DOCUMENT NUMBER: 121:173326
ORIGINAL REFERENCE NO.: 121:31327a,31330a
TITLE: The sequence of porcine apolipoprotein E (APOE) cDNA
AUTHOR(S): Brzozowska, Anna; Grimholt, Unni; Kulseth, Mari Ann; Wold, Inger; Rogne, Sissel
CORPORATE SOURCE: Dep. Anim. Sci., Agricultural Univ., Aas, 1432, Norway
SOURCE: DNA Sequence (1993), 4(3), 207-10
CODEN: DNSEES; ISSN: 1042-5179
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Porcine cDNA clones encoding apolipoprotein E (APOE) were isolated and sequenced. The porcine APOE cDNA sequence is 1122 bp in length and encodes a pre-protein of 317 amino acids. The inferred porcine amino acid sequence corresponds to the human APOE-4 isoform.

L25 ANSWER 21 OF 22 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1992:4175 HCAPLUS Full-text
DOCUMENT NUMBER: 116:4175
ORIGINAL REFERENCE NO.: 116:823a,826a
TITLE: Investigation of a possible correlation between rates of secretion and microsomal membrane association of plasma proteins synthesized by rat liver
AUTHOR(S): Myrset, Astrid Hilde; Johnsen, Beate Rygg; Kulseth, Mari Ann; Wassdal, Irene; Helgeland, Liv
CORPORATE SOURCE: Dep. Biochem., Univ. Oslo, Oslo, 0316, Norway
SOURCE: Biochimica et Biophysica Acta, Biomembranes (1991), 1070(1), 229-36
CODEN: BBBMBS; ISSN: 0005-2736
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The rates of secretion of complement C3, haptoglobin, and plasminogen were determined after pulse labeling with [3H]leucine, and compared to the secretion of prothrombin, albumin, and transferrin investigated previously (Kvalvaag, A. H., et al., 1988). To study membrane association, rough microsomes were treated with increasing concns. of saponin, sodium deoxycholate, or Triton X-100. All 6 proteins were quantitated in the soluble and membrane fraction by enzyme immunoassays. At concns. of saponin of 0.08-0.32%, each secretory protein showed a characteristic distribution, almost identical to that obtained with 0.05% sodium deoxycholate or 0.08% Triton X-100. Albumin and transferrin with half-times for secretion (t1/2) 30 and 75 min, resp., are both almost exclusively found in the luminal fraction (>95%). Prothrombin and plasminogen, which both show an intermediate t1/2 (.apprx.55 min), are partially associated with the membranes, as only .apprx.60% was released. Haptoglobin and complement C3 also show some association with the membranes (80-85% released). C3 is secreted at the same rate as prothrombin and plasminogen (t1/2 = 55 min), whereas haptoglobin is secreted more rapidly (t1/2 = 40 min). Accordingly, no correlation between kinetics of secretion and membrane association was demonstrated.

L25 ANSWER 22 OF 22 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:454331 BIOSIS Full-text
DOCUMENT NUMBER: PREV200400459240
TITLE: Method for the identification of a receptor.
AUTHOR(S): Kulseth, Mari Ann [Inventor, Reprint Author]; Lovhaug, Dagfinn [Inventor]; Godal, Aslak [Inventor]
CORPORATE SOURCE: Oslo, Norway
ASSIGNEE: Amersham Health AS, Oslo, Norway
PATENT INFORMATION: US 6806045 20041019
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Oct 19 2004) Vol. 1287, No. 3. http://www.uspto.gov/web/menu/patdata.html. e-file.
ISSN: 0098-1133 (ISSN print).
DOCUMENT TYPE: Patent
LANGUAGE: English
ENTRY DATE: Entered STN: 24 Nov 2004
Last Updated on STN: 24 Nov 2004
AB A method for the identification and characterization of a receptor in target tissue for which a selected vector has affinity, wherein a transfected cell line expressing the receptor is added to a suspension of encapsulated microbubbles to which the selected vector is coupled and allowing the microbubbles and cells coupled thereto to float to the surface of the suspension. Upon isolating the microbubble-bound cells at the surface, these

may be cultured to study the receptor, or cells may be lysed, amplifying the receptor-encoding cDNA and sequencing the cDNA.

Sequence search history in STN REGISTRY

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L3            2 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L2
L4            1 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L3 AND (HEPARIN OR " VEGF" OR
                "VEGFR-2" OR (VEGFR(W)2) OR (BIND?(2A)(DOMAIN OR REGION OR
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L5            2 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L3 OR L4


=> d his L13

     (FILE 'USPATFULL' ENTERED AT 08:41:32 ON 12 JUN 2008)
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L12           1 SEA FILE=USPATFULL ABB=ON  PLU=ON  L2
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FILE 'HCAPLUS' ENTERED AT 08:53:50 ON 12 JUN 2008
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FILE 'USPATFULL' ENTERED AT 08:53:50 ON 12 JUN 2008
CA INDEXING COPYRIGHT (C) 2008 AMERICAN CHEMICAL SOCIETY (ACS)
PROCESSING COMPLETED FOR L5
PROCESSING COMPLETED FOR L13
L26           3 DUP REM L5 L13 (0 DUPLICATES REMOVED)
                ANSWERS '1-2' FROM FILE HCAPLUS
                ANSWER '3' FROM FILE USPATFULL
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L26  ANSWER 1 OF 3  HCAPLUS  COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER:         2004:566644  HCAPLUS  Full-text
DOCUMENT NUMBER:          141:117188
TITLE:                    Peptides that bind to the heparin-
                          binding domain of VEGF and
                          VEGFR-2, preparation, and
                          therapeutic and diagnostic use
INVENTOR(S):              Kulseth, Mari Ann
PATENT ASSIGNEE(S):       Amersham Health As, Norway
SOURCE:                   PCT Int. Appl., 31 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.           KIND   DATE        APPLICATION NO.         DATE
     ---------------      ----   --------    --------------------    --------
     WO 2004058802         A1    20040715    WO 2003-NO444           20031229
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
             CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
             GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
             LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO,
             NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ,
             TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ,
             BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE,
             ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK,
             TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
     AU 2003288810         A1    20040722    AU 2003-288810          20031229
     EP 1578785            A1    20050928    EP 2003-781116          20031229
     EP 1578785            B1    20080213
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK
     JP 2006523608         T     20061019    JP 2004-563063          20031229
     AT 386050             T     20080315    AT 2003-781116          20031229
     US 20060089307        A1    20060427    US 2005-540431          20050622
PRIORITY APPLN. INFO.:                       NO 2002-6286         A  20021230
                                             WO 2003-NO444        W  20031229

OTHER SOURCE(S):          MARPAT 141:117188
ED   Entered STN:  15 Jul 2004
AB   The invention discloses peptides for targeting that bind to the heparin-
     binding domain of vascular endothelial growth factor and its receptor vascular
     endothelial growth factor receptor 2, VEGFR-2.  The invention also discloses
     the use of these peptides in therapeutically effective treatment as well as
     for diagnostic imaging techniques.  Preparation of peptides is described.
IC   ICM  C07K002-00
     ICS  C07K007-06; C07K007-08; A61K047-48; C07K014-475; C07K014-71
CC   1-12 (Pharmacology)
     Section cross-reference(s): 8, 34
ST   diagnostic imaging peptide prepn VEGF VEGFR2 heparin
     binding domain; therapeutic peptide prepn VEGF
     VEGFR2 heparin binding domain
IT   Imaging agents
        (contrast; peptides binding to heparin-binding
        domain of VEGF and VEGFR-2,
```

preparation, and therapeutic and diagnostic use)

IT Metals, biological studies
RL: DGN (Diagnostic use); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(paramagnetic and fluorescent, peptide conjugates; peptides binding to heparin-binding domain of VEGF and VEGFR-2, preparation, and therapeutic and diagnostic use)

IT Antitumor agents
Cluster ions
Fluorescent substances
Paramagnetic materials
(peptide conjugates; peptides binding to heparin-binding domain of VEGF and VEGFR-2, preparation, and therapeutic and diagnostic use)

IT Heavy metals
Radionuclides, biological studies
RL: DGN (Diagnostic use); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(peptide conjugates; peptides binding to heparin-binding domain of VEGF and VEGFR-2, preparation, and therapeutic and diagnostic use)

IT Diagnosis
Drug delivery systems
Drug targets
Human
Imaging agents
Linking agents
Neoplasm
(peptides binding to heparin-binding domain of VEGF and VEGFR-2, preparation, and therapeutic and diagnostic use)

IT Peptides, biological studies
RL: DGN (Diagnostic use); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(peptides binding to heparin-binding domain of VEGF and VEGFR-2, preparation, and therapeutic and diagnostic use)

IT Vascular endothelial growth factor receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(type VEGFR-2; peptides binding to heparin-binding domain of VEGF and VEGFR-2, preparation, and therapeutic and diagnostic use)

IT 13981-59-4, Tin-117, biological studies 14133-76-7, Technetium-99, biological studies
RL: DGN (Diagnostic use); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(metastable, peptide conjugates; peptides binding to heparin-binding domain of VEGF and VEGFR-2, preparation, and therapeutic and diagnostic use)

IT 9005-49-6, Heparin, biological studies 127464-60-2, Vascular endothelial growth factor
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(peptides binding to heparin-binding domain of VEGF and VEGFR-2, preparation, and therapeutic and diagnostic use)

IT 721887-16-7P 721887-21-4P
RL: DGN (Diagnostic use); PAC (Pharmacological activity); PRP (Properties); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)

(peptides binding to heparin-binding domain of VEGF and VEGFR-2, preparation, and therapeutic and diagnostic use)

IT 721886-53-9 721886-57-3 721886-62-0 721886-65-3 721886-69-7 721886-71-1 721886-75-5 721886-79-9 721886-83-5 721886-87-9 721886-91-5 721886-96-0 721887-01-0 721887-06-5 722580-42-9 722580-43-0

RL: DGN (Diagnostic use); PAC (Pharmacological activity); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(peptides binding to heparin-binding domain of VEGF and VEGFR-2, preparation, and therapeutic and diagnostic use)

IT 50-18-0D, Cyclophosphamide, peptide conjugates 51-21-8D, Fluorouracil, peptide conjugates 55-98-1D, Busulfan, peptide conjugates 59-05-2D, Methotrexate, peptide conjugates 147-94-4D, Cytarabine, peptide conjugates 305-03-3D, Chlorambucil, peptide conjugates 865-21-4D, Vinblastine, peptide conjugates 10098-91-6D, Yttrium-90, peptide conjugates, biological studies 13967-74-3D, Cerium-141, peptide conjugates, biological studies 13981-56-1D, Fluorine-18, peptide conjugates, biological studies 14119-09-6D, Gallium-67, peptide conjugates, biological studies 14265-75-9D, Lutetium-177, peptide conjugates, biological studies 14391-11-8D, Gold-199, peptide conjugates, biological studies 14391-22-1D, Thulium-167, peptide conjugates, biological studies 14391-96-9D, Scandium-47, peptide conjugates, biological studies 14392-02-0D, Chromium-51, peptide conjugates, biological studies 14687-25-3D, Lead-203, peptide conjugates, biological studies 14998-63-1D, Rhenium-186, peptide conjugates, biological studies 15663-27-1D, Cisplatin, peptide conjugates 15750-15-9D, Indium-111, peptide conjugates, biological studies 15757-86-5D, Copper-67, peptide conjugates, biological studies 15758-35-7D, Ruthenium-97, peptide conjugates, biological studies 20830-81-3D, Daunorubicin, peptide conjugates 23214-92-8D, Doxorubicin, peptide conjugates 29767-20-2D, Teniposide, peptide conjugates 33069-62-4D, Paclitaxel, peptide conjugates 33419-42-0D, Etoposide, peptide conjugates 51264-14-3D, Amsacrine, peptide conjugates 114977-28-5D, Docetaxel, peptide conjugates

RL: DGN (Diagnostic use); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(peptides binding to heparin-binding domain of VEGF and VEGFR-2, preparation, and therapeutic and diagnostic use)

IT 721887-11-2P

RL: PRP (Properties); RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)

(peptides binding to heparin-binding domain of VEGF and VEGFR-2, preparation, and therapeutic and diagnostic use)

IT 721887-21-4P

RL: DGN (Diagnostic use); PAC (Pharmacological activity); PRP (Properties); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)

(peptides binding to heparin-binding domain of VEGF and VEGFR-2, preparation, and therapeutic and diagnostic use)

RN 721887-21-4 HCAPLUS

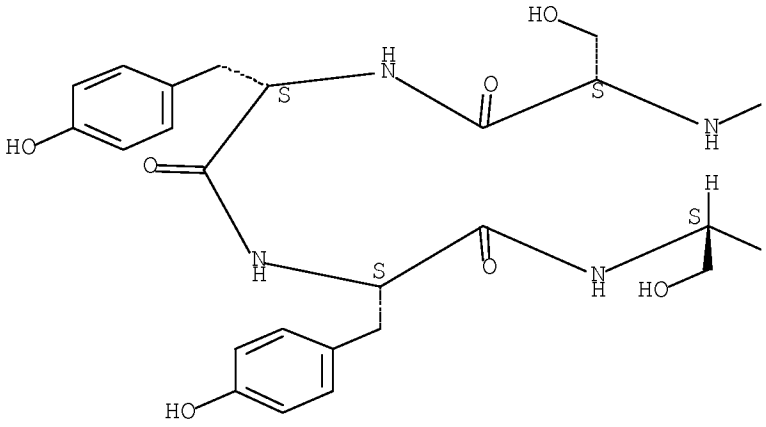
CN L-Cysteinamide, L-cysteinyl-L-seryl-L-tyrosyl-L-tyrosyl-L-seryl-L-α-aspartylglycyl-L-valyl-L-tyrosyl-L-α-aspartyl-L-cysteinylglycyl-, cyclic (1→11)-disulfide (9CI) (CA INDEX NAME)

NTE modified

SEQ 1 CSYYSDGVYD CGC

Absolute stereochemistry.

PAGE 1-A



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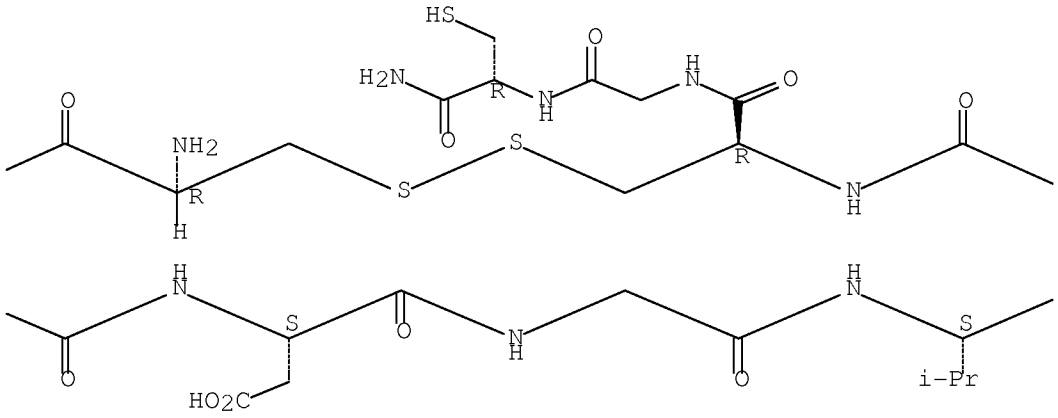

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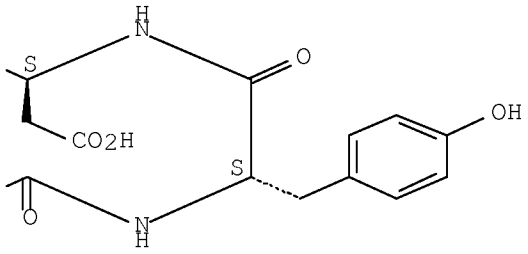


IT 721886-53-9 721886-57-3 721886-62-0
721886-65-3 721886-69-7 721886-71-1
721886-75-5 721886-83-5 721886-96-0
721887-06-5
RL: DGN (Diagnostic use); PAC (Pharmacological activity); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(peptides binding to heparin-binding domain of VEGF and VEGFR-2, preparation, and therapeutic and diagnostic use)

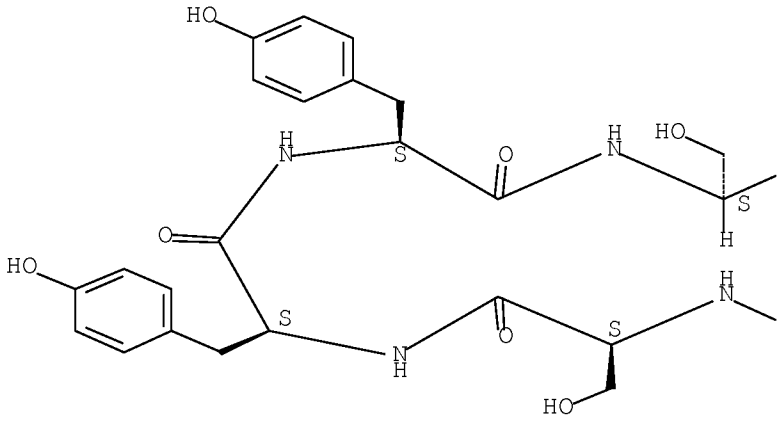
RN 721886-53-9 HCAPLUS

CN L-Cysteine, L-cysteinyl-L-seryl-L-tyrosyl-L-tyrosyl-L-seryl-L-α-aspartylglycyl-L-valyl-L-tyrosyl-L-α-aspartyl-, cyclic (1→11)-disulfide (9CI) (CA INDEX NAME)

SEQ 1 CSYYSDGVYD C

Absolute stereochemistry.

PAGE 1-A



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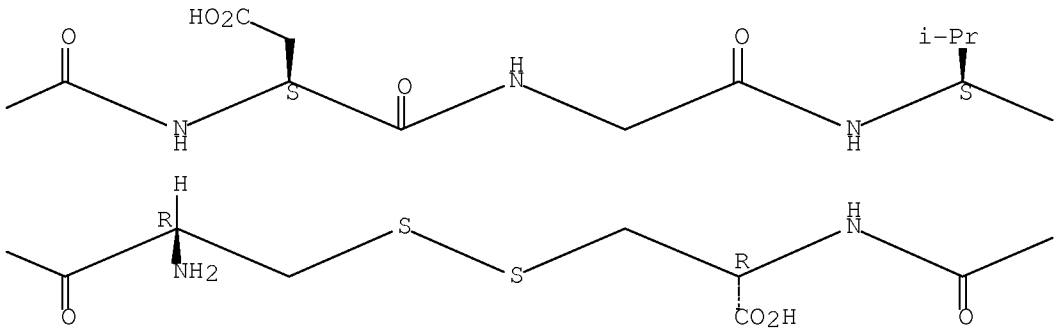

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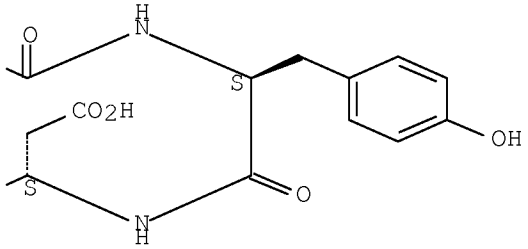


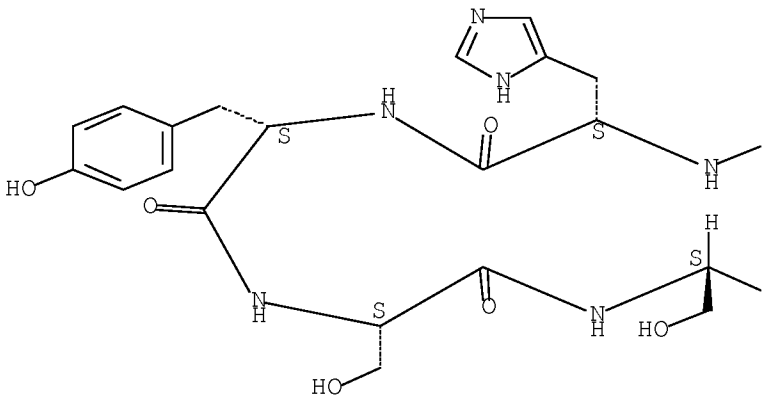
RN 721886-57-3 HCAPLUS

CN L-Cysteine, L-cysteinyl-L-histidyl-L-tyrosyl-L-seryl-L-seryl-L-α-aspartylglycyl-L-threonyl-L-tyrosyl-L-α-aspartyl-, cyclic (1→11)-disulfide (9CI) (CA INDEX NAME)

SEQ 1 CHYSSDGTYD C

Absolute stereochemistry.

PAGE 1-A



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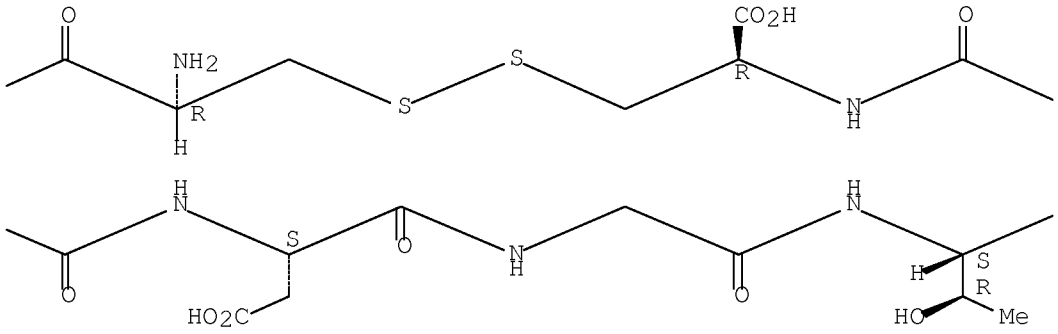

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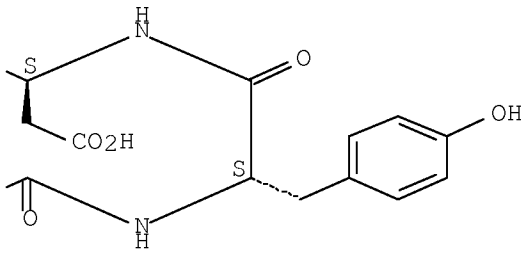


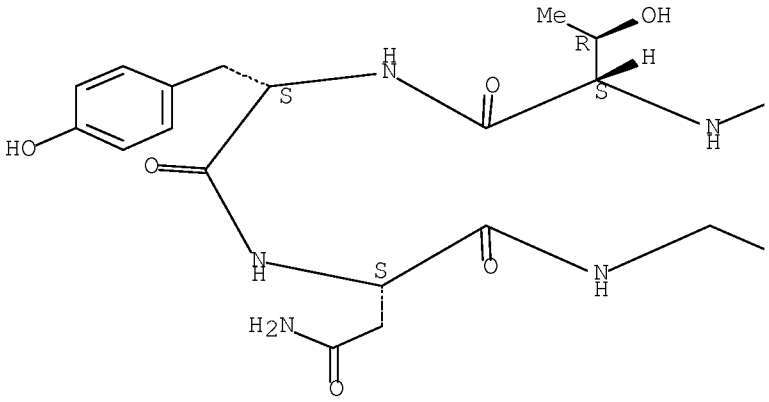
RN 721886-62-0 HCAPLUS

CN L-Cysteine, L-cysteinyl-L-threonyl-L-tyrosyl-L-asparaginylglycyl-L-α-aspartylglycyl-L-seryl-L-phenylalanyl-L-α-aspartyl-, cyclic (1→11)-disulfide (9CI) (CA INDEX NAME)

SEQ 1 CTYNGDGSFD C

Absolute stereochemistry.

PAGE 1-A



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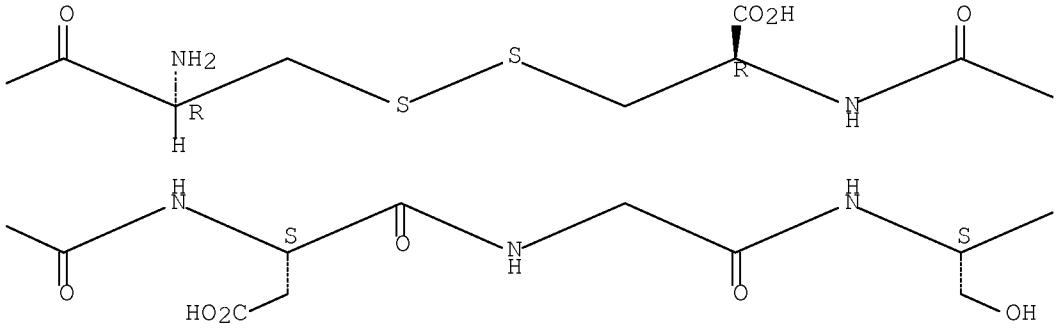

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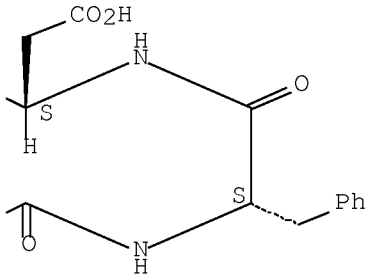


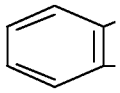
RN 721886-65-3 HCAPLUS

CN L-Cysteine, L-cysteinyl-L-alanyl-L-tyrosyl-L-α-glutamyl-L-alanyl-L-α-aspartylglycyl-L-tryptophyl-L-phenylalanyl-L-α-aspartyl-, cyclic (1→11)-disulfide (9CI) (CA INDEX NAME)

SEQ 1 CAYEADGWFD C

Absolute stereochemistry.

PAGE 1-A



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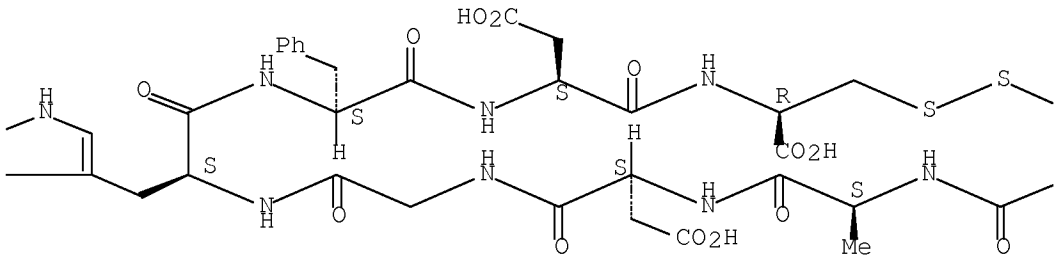

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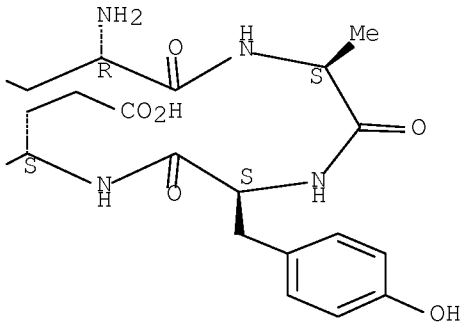


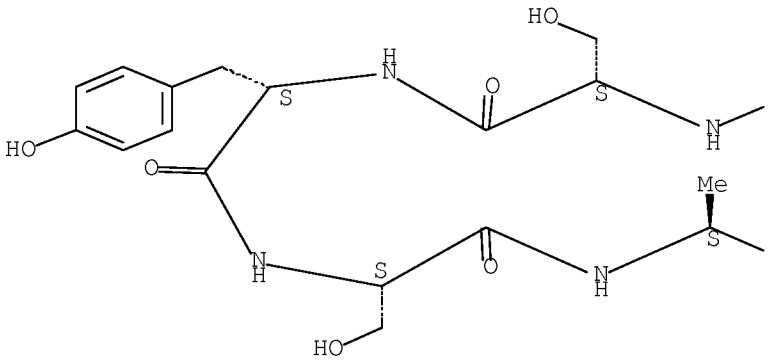
RN 721886-69-7 HCAPLUS

CN L-Cysteine, L-cysteinyl-L-seryl-L-tyrosyl-L-seryl-L-alanyl-L-α-aspartylglycyl-L-threonyl-L-leucyl-L-α-aspartyl-, cyclic (1→11)-disulfide (9CI) (CA INDEX NAME)

SEQ 1 CSYSADGTLD C

Absolute stereochemistry.

PAGE 1-A



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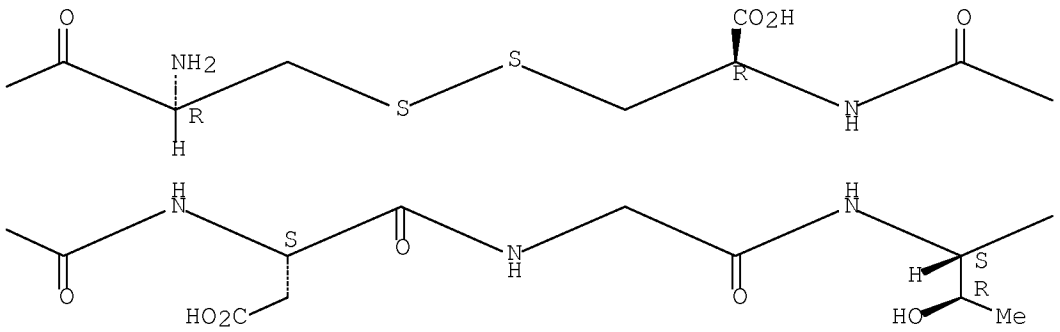

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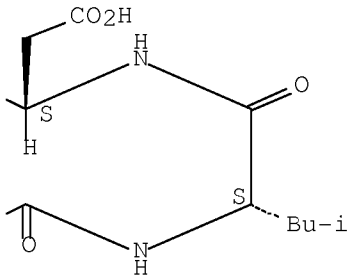


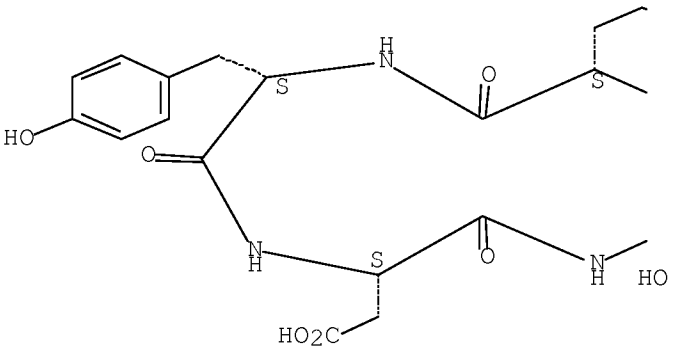
RN 721886-71-1 HCAPLUS

CN L-Cysteine, L-cysteinyl-L-glutaminyl-L-tyrosyl-L-α-aspartyl-L-seryl-L-serylglycyl-L-methionyl-L-tyrosyl-L-α-aspartyl-, cyclic (1→11)-disulfide (9CI) (CA INDEX NAME)

SEQ 1 CQYDSSGMYD C

Absolute stereochemistry.

PAGE 1-A



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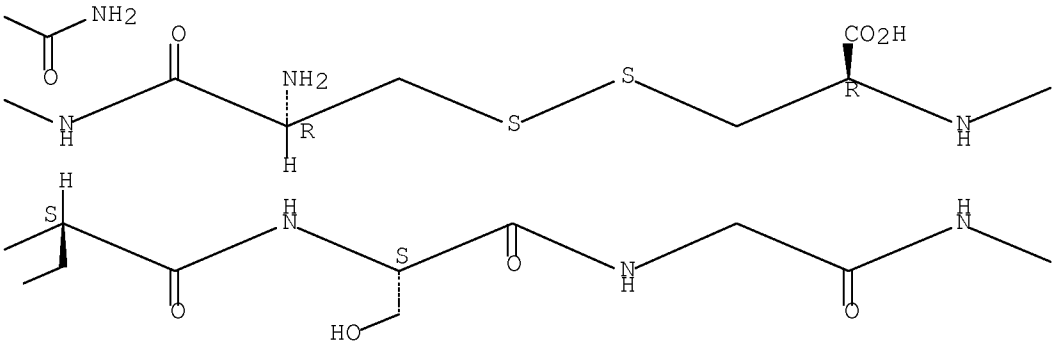

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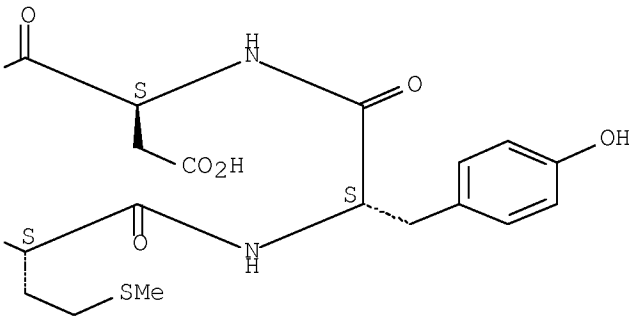


RN 721886-75-5 HCAPLUS

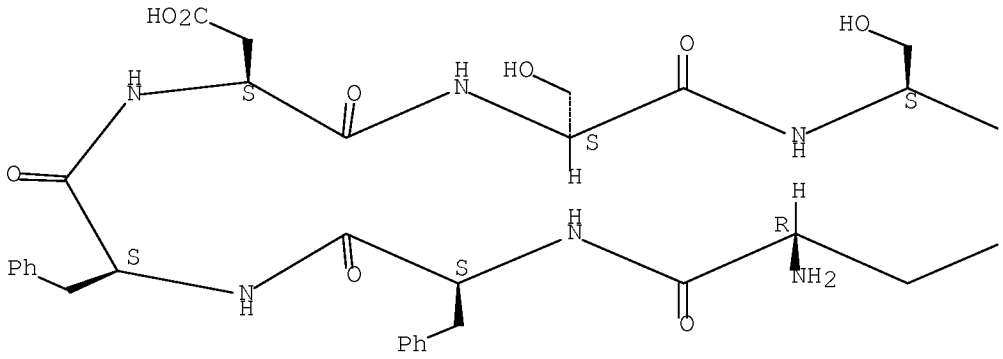
CN L-Cysteine, L-cysteinyl-L-phenylalanyl-L-phenylalanyl-L-α-aspartyl-L-

seryl-L-serylglycyl-L-tyrosyl-L-phenylalanyl-L-α-aspartyl-, cyclic (1→11)-disulfide (9CI) (CA INDEX NAME)

SEQ 1 CFFDSSGYFD C

Absolute stereochemistry.

PAGE 1-A



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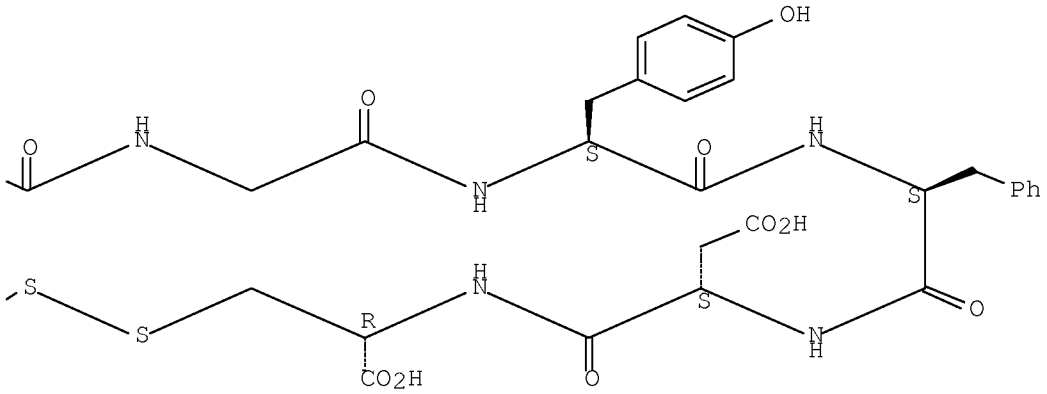


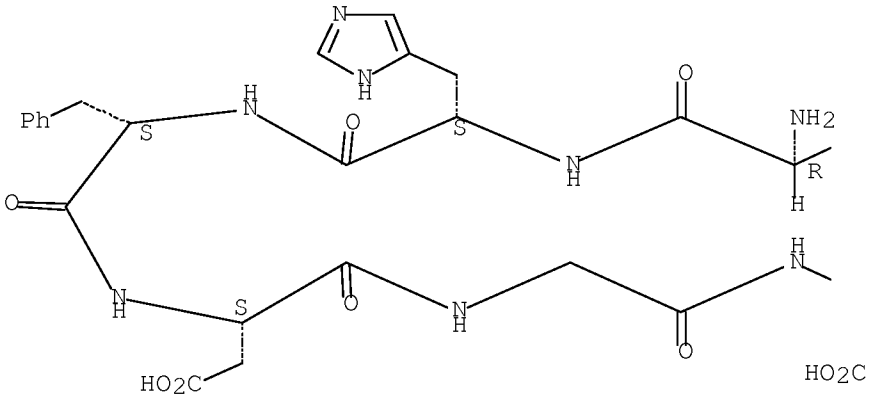
RN 721886-83-5 HCAPLUS

CN L-Cysteine, L-cysteinyl-L-histidyl-L-phenylalanyl-L-α-aspartylglycyl-L-α-aspartylglycyl-L-seryl-L-tyrosyl-L-α-aspartyl-, cyclic (1→11)-disulfide (9CI) (CA INDEX NAME)

SEQ 1 CHFDGDGSYD C

Absolute stereochemistry.

PAGE 1-A



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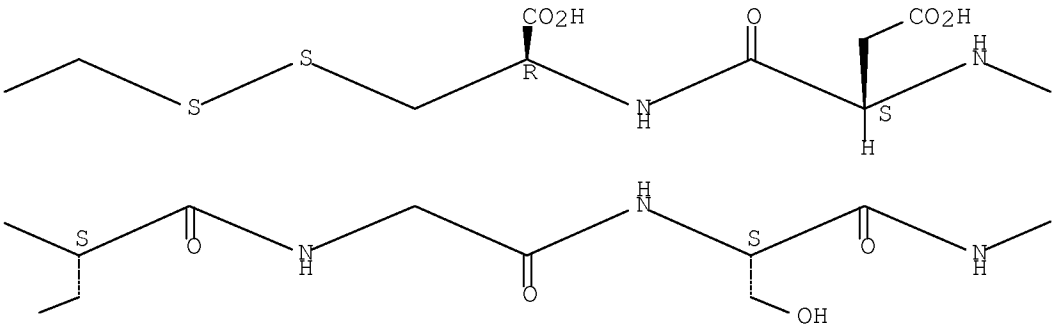

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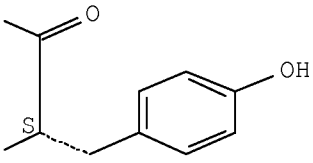

RN 721886-96-0 HCAPLUS

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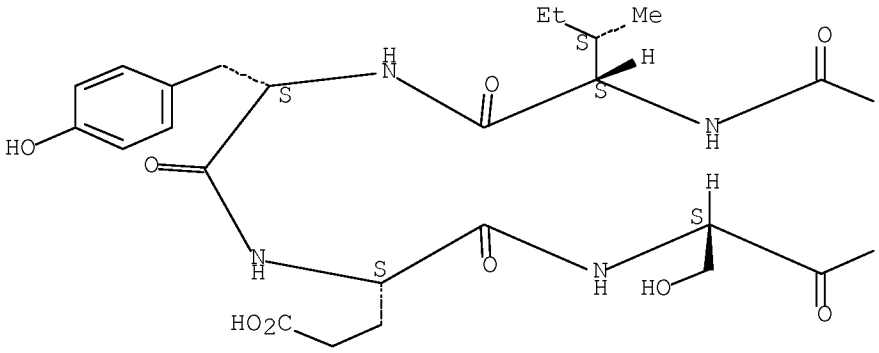
SEQ 1 CIYESDGMFS C

Absolute stereochemistry.

PAGE 1-A



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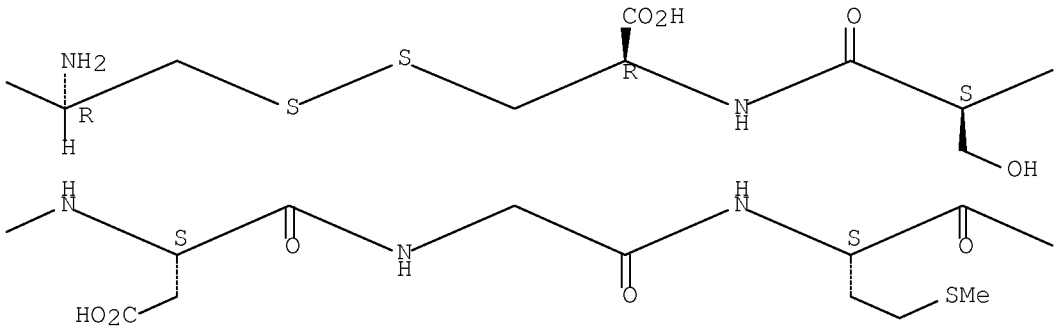

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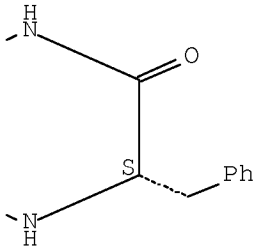

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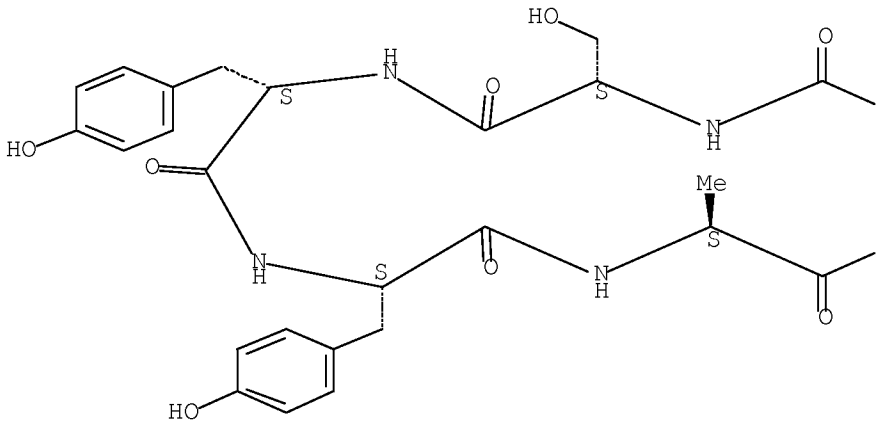
SEQ 1 CSYYADGMYS C

Absolute stereochemistry.

PAGE 1-A



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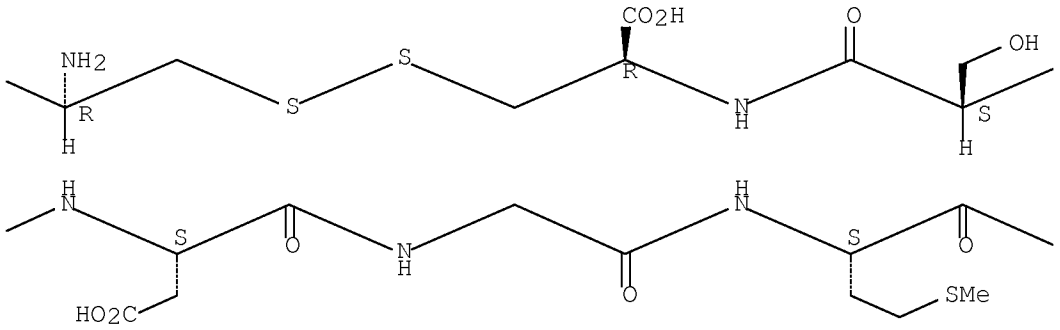

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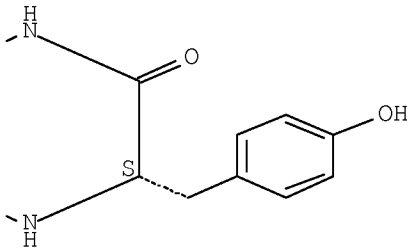

L26 ANSWER 2 OF 3 HCAPLUS COPYRIGHT 2008 ACS on STN
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AUTHOR(S): Moran, Mary Ann; Buchan, Alison; Gonzalez, Jose M.; Heidelberg, John F.; Whitman, William B.; Kiene, Ronald P.; Henriksen, James R.; King, Gary M.; Belas, Robert; Fuqua, Clay; Brinkac, Lauren; Lewis, Matt; Johri, Shivani; Weaver, Bruce; Pai, Grace; Eisen, Jonathan A.; Rahe, Elisha; Sheldon, Wade M.; Ye, Wenying; Miller, Todd R.; Carlton, Jane; Rasko, David A.; Paulsen, Ian T.; Ren, Qinghu; Daugherty, Sean C.; Deboy, Robert T.; Dodson, Robert J.; Durkin, A. Scott; Madupu, Ramana; Nelson, William C.; Sullivan, Steven A.; Rosovitz, M. J.; Haft, Daniel H.; Selengut, Jeremy; Ward, Naomi
CORPORATE SOURCE: Department of Marine Sciences and, Dep. Marine SCiences, Univ. Georgia, Athens, GA, 30602, USA
SOURCE: Nature (London, United Kingdom) (2004), 432(7019), 910-913
CODEN: NATUAS; ISSN: 0028-0836
PUBLISHER: Nature Publishing Group

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LANGUAGE: English
ED Entered STN: 16 Dec 2004
AB Since the recognition of prokaryotes as essential components of the oceanic food web, bacterioplankton have been acknowledged as catalysts of most major biogeochem. processes in the sea. Studying heterotrophic bacterioplankton has been challenging, however, as most major clades have never been cultured or have only been grown to low densities in sea water. This report describes the genome sequence of Silicibacter pomeroyi, a member of the marine Roseobacter clade, the relatives of which comprise .apprx.10-20% of coastal and oceanic mixed-layer bacterioplankton. This first genome sequence from any major heterotrophic clade consists of a chromosome (4,109,442 base pairs) and megaplasmid (491,611 base pairs). Genome anal. indicates that this organism relies upon a lithoheterotrophic strategy that uses inorg. compds. (carbon monoxide and sulfide) to supplement heterotrophy. Silicibacter pomeroyi also has genes advantageous for assocns. with plankton and suspended particles, including genes for uptake of algal-derived compds., use of metabolites from reducing microzones, rapid growth and cell-d.-dependent regulation. This bacterium has a physiol. distinct from that of marine oligotrophs, adding a new strategy to the recognized repertoire for coping with a nutrient-poor ocean. The complete sequence has been submitted to GenBank/EMBL/DDBJ under accession nos. CP000031 and CP000032.
CC 3-3 (Biochemical Genetics)
Section cross-reference(s): 6, 10
IT 800275-71-2 800275-72-3 800275-73-4 800275-74-5 800275-75-6
800275-76-7 800275-77-8 800275-78-9 800275-79-0 800275-80-3
800275-81-4 800275-82-5 800275-83-6 800275-84-7 800275-85-8
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800278-05-1 800278-06-2 800278-07-3
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(amino acid sequence; genome sequence of Silicibacter pomeroyi reveals adaptations to the marine environment)

IT 800276-18-0
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(amino acid sequence; genome sequence of Silicibacter pomeroyi reveals adaptations to the marine environment)

RN 800276-18-0 HCAPLUS
CN Protein (Silicibacter pomeroyi strain DSS-3 558-amino acid) (9CI) (CA INDEX NAME)

SEQ
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  1 MMFNTLISIG AASRRVRNRL KDRANAFARE EDGLMTVMAL FLFLALVGAA
 51 GIGVDLMRYE QKRAALQYTM DRAVLAAADL DQQVSPETVV RSYLEKAGLL
101 EYLSSVTVQE GLGYRKVSAT ATAELPTHFM KLSGYDSLTI PAASTAEESI
151 GNVEISLVLD VSGSMNSNSR LYNLKNAAKE FVDHMLSATE PGTVSISIVP
201 YATQVNAGAD ILSYYNVSTE HNYSHCVNFI DDEFSQPGLS RVTPLERTMH
251 FDPFSYTKDP ISTPVCPVRA STEILPFSND QTVLNNYIDG LTGRGNTSID
301 IGTKWGVVML DPGTQSVISG LISDNKVPAS FQGRPSAYDS GDVLKVLIVM
351 SDGENTNQYM LNPSLRDGDS PVWYNAAEDV ISGSPDNNTT NAFSIYHDNG
401 NNSYYWPDQN RWADHPYGNG QSEACGYNSS GYYSCAMRDE PGEAVRLTYA
451 ELFAKVSLAY NAYYNFEFNS NAWAEWYTAA MTHKEASAKD QRTDHVCDAA
501 KDEGIIVYTV GFEAPYSGRR VLKRCASSDS HYYDADGLEI SDAFTSIASS
551 IRKLRLTQ
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REFERENCE COUNT: 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d L26 3 ibib ab hit

L26 ANSWER 3 OF 3 USPATFULL on STN

ACCESSION NUMBER:	2006:105199 USPATFULL	Full-text
TITLE:	Peptides that bind to the heparin binding domian of vegf and vegfr-2	
INVENTOR(S):	Kulseth, Mari Ann, Oslo, NORWAY	

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 20060089307	A1	20060427	
APPLICATION INFO.:	US 2003-540431	A1	20031229	(10)
	WO 2003-NO444		20031229	
			20050622	PCT 371 date

	NUMBER	DATE
PRIORITY INFORMATION:	NO 2002-6286	20021230

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: AMERSHAM HEALTH, IP DEPARTMENT, 101 CARNEGIE CENTER, PRINCETON, NJ, 08540-6231, US
NUMBER OF CLAIMS: 10
EXEMPLARY CLAIM: 1
LINE COUNT: 735
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to new peptides for targeting that bind to the heparin binding domain of vascular endothelial growth factor and its receptor vascular endothelial growth factor receptor 2, VEGFR-2. The invention further relates to their use in therapeutically effective treatment as well as for diagnostic imaging techniques.

IT 721887-16-7P 721887-21-4P
(peptides binding to heparin-binding domain of VEGF and VEGFR-2, preparation, and therapeutic and diagnostic use)

IT 721886-53-9 721886-57-3 721886-62-0
721886-65-3 721886-69-7 721886-71-1
721886-75-5 721886-79-9 721886-83-5 721886-87-9
721886-91-5 721886-96-0 721887-01-0 721887-06-5
722580-42-9 722580-43-0
(peptides binding to heparin-binding domain of VEGF and VEGFR-2, preparation, and therapeutic and diagnostic use)

Full search history

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=> d his full

     (FILE 'HOME' ENTERED AT 08:26:34 ON 12 JUN 2008)

     FILE 'HCAPLUS' ENTERED AT 08:28:35 ON 12 JUN 2008
                E US20060089307 /PN
L1              1 SEA ABB=ON  PLU=ON  US20060089307 /PN
                D L1
                D SCAN

     FILE 'REGISTRY' ENTERED AT 08:29:28 ON 12 JUN 2008
L2             12 SEA ABB=ON  PLU=ON  [C'HCY'][SHTAQFGI][YRF][YSNEDT][SAGDF][DS]G
                [TVMSWY][YFL][DSE][C'HCY']/SQSP

     FILE 'HCAPLUS' ENTERED AT 08:32:32 ON 12 JUN 2008
L3              2 SEA ABB=ON  PLU=ON  L2
                D L3 1-2 TI
                D L3 1-2 AU
                D SCAN
L4              1 SEA ABB=ON  PLU=ON  L3 AND (HEPARIN OR " VEGF" OR "VEGFR-2" OR
                (VEGFR(W)2) OR (BIND?(2A)(DOMAIN OR REGION OR RECEPT?)))
L5              2 SEA ABB=ON  PLU=ON  L3 OR L4
                E KULSETH M?/AU
L6             21 SEA ABB=ON  PLU=ON  ("KULSETH M A"/AU OR "KULSETH MARI A"/AU
                OR "KULSETH MARI ANN"/AU)
L7              9 SEA ABB=ON  PLU=ON  L6 AND GE?/CO,CS,PA,SO
L8              2 SEA ABB=ON  PLU=ON  L6 AND HEALTH?/CO,CS,PA,SO
L9              2 SEA ABB=ON  PLU=ON  L6 AND (HEPARIN OR "VEGF" OR "VEGFR-2" OR
                (VEGFR(W)2) OR (PEPTID?(3A)(BIND? OR DOMAIN OR REGION)))
L10            21 SEA ABB=ON  PLU=ON  (L6 OR L7 OR L8 OR L9)
                D L10 1-21 AU
                D L10 1-21 AU
                SAVE TEMP L5 HEA431HCSQ/A
                SAVE TEMP L10 HEA431HCIN/A

     FILE 'USPATFULL' ENTERED AT 08:41:32 ON 12 JUN 2008
L11             1 SEA ABB=ON  PLU=ON  L2
L12             1 SEA ABB=ON  PLU=ON  L2
L13             1 SEA ABB=ON  PLU=ON  L11 OR L12
                D L13 TI
                D L13 AU

     FILE 'MEDLINE, BIOSIS, EMBASE' ENTERED AT 08:42:28 ON 12 JUN 2008
L14             0 SEA ABB=ON  PLU=ON  L2
L15             0 SEA ABB=ON  PLU=ON  L3

     FILE 'MEDLINE, BIOSIS, EMBASE, DRUGU' ENTERED AT 08:43:24 ON 12 JUN 2008
L16            50 SEA ABB=ON  PLU=ON  L6
L17            27 SEA ABB=ON  PLU=ON  L7
L18             1 SEA ABB=ON  PLU=ON  L8
L19             1 SEA ABB=ON  PLU=ON  L9
                D L16 1-22 AU
                D L16 1-22 TI
L20             2 SEA ABB=ON  PLU=ON  L16 AND (HEPARIN OR PEPTID? OR ANALOG?)
L21             0 SEA ABB=ON  PLU=ON  L16 AND ("VEGF" OR "VEGFR-2" OR (VEGFR(W)
                2))
L22             1 SEA ABB=ON  PLU=ON  L16 AND AMERSHAM?/CO,CS,PA,SO
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L23           0 SEA ABB=ON  PLU=ON  L16 AND (BIND?(3N)(DOMAIN OR REGION OR
                SITE OR EPITOP?))
L24          30 SEA ABB=ON  PLU=ON  (L17 OR L18 OR L19 OR L20 OR L21 OR L22 OR
                L23)
                SAVE TEMP L24 HEA431MLIN/A

     FILE 'USPATFULL' ENTERED AT 08:50:54 ON 12 JUN 2008
                SAVE TEMP L13 HEA431MLSQ/A
                D QUE L10
                D QUE L24

     FILE 'HCAPLUS, MEDLINE, BIOSIS, EMBASE' ENTERED AT 08:52:20 ON 12 JUN 2008
L25          22 DUP REM L10 L24 (29 DUPLICATES REMOVED)
                     ANSWERS '1-21' FROM FILE HCAPLUS
                     ANSWER '22' FROM FILE BIOSIS
                D L25 1-22 IBIB AB
                D QUE L5
                D QUE L13

     FILE 'HCAPLUS, USPATFULL' ENTERED AT 08:53:50 ON 12 JUN 2008
L26           3 DUP REM L5 L13 (0 DUPLICATES REMOVED)
                     ANSWERS '1-2' FROM FILE HCAPLUS
                     ANSWER '3' FROM FILE USPATFULL
                D L26 1-2 IBIB ED ABS HITIND HITSEQ
                D L26 3 IBIB AB HIT


     FILE HOME

     FILE HCAPLUS

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     FILE COVERS 1907 - 12 Jun 2008  VOL 148 ISS 24
     FILE LAST UPDATED: 11 Jun 2008  (20080611/ED)

     New CAS Information Use Policies, enter HELP USAGETERMS for details.

      This file contains CAS Registry Numbers for easy and accurate
      substance identification.

     FILE REGISTRY
     Property values tagged with IC are from the ZIC/VINITI data file
     provided by InfoChem.

     STRUCTURE FILE UPDATES:   11 JUN 2008  HIGHEST RN 1027436-61-8
     DICTIONARY FILE UPDATES:  11 JUN 2008  HIGHEST RN 1027436-61-8

     New CAS Information Use Policies, enter HELP USAGETERMS for details.

     TSCA INFORMATION NOW CURRENT THROUGH January 9, 2008.
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http://www.cas.org/support/stngen/stndoc/properties.html

FILE USPATFULL
FILE COVERS 1971 TO PATENT PUBLICATION DATE: 10 Jun 2008 (20080610/PD)
FILE LAST UPDATED: 10 Jun 2008 (20080610/ED)
HIGHEST GRANTED PATENT NUMBER: US7386892
HIGHEST APPLICATION PUBLICATION NUMBER: US20080134401
CA INDEXING IS CURRENT THROUGH 10 Jun 2008 (20080610/UPCA)
ISSUE CLASS FIELDS (/INCL) CURRENT THROUGH: 10 Jun 2008 (20080610/PD)
REVISED CLASS FIELDS (/NCL) LAST RELOADED: Apr 2008
USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Apr 2008

FILE MEDLINE
FILE LAST UPDATED: 11 Jun 2008 (20080611/UP). FILE COVERS 1949 TO DATE.

MEDLINE has been updated with the National Library of Medicine's revised 2008 MeSH terms. See HELP RLOAD for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

See HELP RANGE before carrying out any RANGE search.

FILE BIOSIS
FILE COVERS 1926 TO DATE.
CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT
FROM JANUARY 1926 TO DATE.

RECORDS LAST ADDED: 11 June 2008 (20080611/ED)

BIOSIS has been augmented with 1.8 million archival records from 1926 through 1968. These records have been re-indexed to match current BIOSIS indexing.

FILE EMBASE
FILE COVERS 1974 TO 11 Jun 2008 (20080611/ED)

EMBASE was reloaded on March 30, 2008.

EMBASE is now updated daily. SDI frequency remains weekly (default) and biweekly.

This file contains CAS Registry Numbers for easy and accurate substance identification.

Beginning January 2008, Elsevier will no longer provide EMTREE codes as part of the EMTREE thesaurus in EMBASE. Please update your current-awareness alerts (SDIs) if they contain EMTREE codes.

For further assistance, please contact your local helpdesk.

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FILE DRUGU
FILE LAST UPDATED: 9 JUN 2008       <20080609/UP>
>>>  DERWENT DRUG FILE (SUBSCRIBER)  <<<

>>>  FILE COVERS 1983 TO DATE  <<<
>>>  THESAURUS AVAILABLE IN /CT  <<<

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